Care and management of these patients has remained a problem, but there is now increasing interest in the scintigraphic detection of gastrointestinal bleeding. The use of technetium Tc 99m sulfur colloid for the scintigraphic detection of gastrointestinal bleeding has largely been superceded by blood pool imaging.

Winzelberg and colleagues proposed using ^{99m}Tc-tagged red blood cells as a blood pool tracer for detecting gastrointestinal bleeding. Any site actively bleeding within a 24 to 36 hour interval is potentially detectable. Most published series give a sensitivity and specificity for this procedure of about 90%. Bleeding rates of 0.2 to 0.4 ml per minute can be detected. Sequential one-minute views of the abdomen following tracer injection can be stored on computer and displayed in cine mode. This technique allows computerized subtraction of activity in known large blood vessels and other vascular structures. By following the cine display, one may detect the position of extravasated blood at an active bleeding site. A negative study result may obviate the need for emergency angiography and using contrast in patients who are unstable and often elderly.

^{99m}Tc as pertechnetate—not complexed to erythrocytes—can be used to locate ectopic gastric mucosa: Meckel's diverticulum and Barrett's esophagus. Meckel's diverticulum is the most common cause of gastrointestinal bleeding in children younger than 3 years. In a large series of predominantly pediatric patients, Sfakianakis and Conway found that the scanning procedure had a sensitivity of 85%, a specificity of 95%, and an accuracy of 90% for surgically proved ectopic gastric mucosa.

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Emergency Nuclear Medicine Studies

IN RECENT YEARS nuclear medicine studies have played an ever-increasing role in the emergency setting. Several developments have contributed to this change: nuclear medicine services are more widely available, new radiotracers have become available, image quality has improved, and radiopharmaceuticals are easier to prepare.

Lung scans have been a standard test for detecting acute pulmonary emboli for many years. More recently, radionuclide biliary studies are commonly used in detecting cystic duct obstruction in acute cholecystitis, common duct obstruction, or bile leak. Tagged red blood cell studies are used to detect and localize acute gastrointestinal bleeding and radionuclide venograms to identify acute thrombophlebitis and superior vena cava obstruction.

In addition, a liver-spleen scan is useful in diagnosing fracture, laceration, and hematoma; renal studies in renal artery obstruction and acute renal failure; testicular studies in torsion and acute epididymitis; pyrophosphate cardiac studies in acute myocardial infarction and cardiac contusion; and brain scans in acute viral encephalitis and for determining brain death. The early determination of brain death has taken on increasing importance because of organ transplant programs.

Emergency bone scans may show fractures missed on radiographs because separation has not yet occurred. They can provide an overview of the entire skeleton in a possibly battered child. Bone scans are also helpful in detecting stress fracture, early acute osteomyelitis, septic arthritis, and avascular necrosis.

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A Simple Bedside Method for the Prompt Diagnosis of Brain Death

BECAUSE BRAIN FUNCTION is the essence of human life, death of this organ is recognized as a criterion of death. Indeed, attempts at resuscitation following cardiac arrest are aimed at sustaining the brain. The Harvard criteria, established in 1968, require 24 hours of electrocerebral silence in appropriate comatose patients in the absence of drug intoxication or hypothermia. A totally flat electroencephalogram is often difficult to obtain reliably. Screening for a variety of drugs required by these criteria is time-consuming and may delay diagnosis. While undue delay is most critical in the case of potential organ donors, a futile attempt at maintaining life in a brain-dead person is very costly and often causes unnecessary anguish for relatives.

Demonstration of a deficit in blood flow to the brain is diagnostic of brain death and is not related to the cause of coma. This absence of circulation can be shown angiographically. There is, however, a justifiable reluctance to moving these fragile, comatose patients, who may or may not be brain dead, from their bedside life support systems to submit them to angiography when they will not benefit directly from this procedure. A critical deficit in cerebral blood flow can be reliably shown by nuclear imaging procedures. Using a portable γ camera, the study can be done at a patient's bedside. The cerebral transit of an intravenously injected bolus of an agent such as technetium Tc 99m pertechnetate, pentetic acid (DTPA), or glucoheptonate can be followed by rapid sequential imaging of the head. This dynamic sequence will reliably show the absence of intracerebral blood flow. A static anterior image of the head following the flow study will, in the majority of patients, be confirmatory by showing an absence of activity in the sagittal sinus. In a small number of patients, the sagittal sinus may be visualized even in the absence of brain activity. This is presumably due to ingress of the imaging agent through the emissary veins. This in no way negates an abnormal flow study result.

This nuclear medicine study is essentially free of risk, simple, and absolutely reliable—except in infants. The study may be repeated for further confirmation or if the first examination was technically unsatisfactory. The more up-to-date and rapid diagnostic criteria for the diagnosis of brain death

are included in the recommendation of the consultants on the President's Commission on the Diagnosis of Death. The nuclear medicine test is recommended as confirmatory by these consultants.

It is worth noting that this test does not evaluate the posterior fossa. Other tests are available for this and, in any case, in the presence of total supratentorial brain death, demise of the posterior fossa will occur within a relatively few hours.

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Simple Evaluation of Thyroid Function

PHYSIOLOGIC AND CLINICAL STUDIES have shown clearly that primary thyroid failure is associated with a compensatory rise in pituitary thyroid-stimulating hormone (TSH) secretion, while an excess of circulating thyroid hormones is associated with a negative feedback inhibition of TSH secretion. Until very recently commercial assays for TSH could detect abnormally high serum levels but were not sensitive enough to distinguish abnormally low values from those found in most normal persons. Consequently, the TSH assay was used primarily to confirm or establish the diagnosis of hypothyroidism. Less commonly, the failure of the serum TSH to rise in response to the administration of thyrotropin-releasing hormone was used to confirm the diagnosis of hyperthyroidism in clinically equivocal cases.

The advent of the ultrasensitive test for thyroid-stimulating hormone, which is capable of detecting levels in serum below those found in normal subjects, is revolutionizing the laboratory assessment of thyroid function. All forms of hyperthyroidism, with the exception of the rare TSH-secreting pituitary adenoma—Graves' disease, toxic thyroid adenoma, Hashimoto's thyroiditis, subacute thyroiditis, or excessive ingestion of thyroxine or triiodothyronine—are associated with serum TSH values well below the defined and measurable normal range of about 0.4 to 6.2 μU per ml. The new type of TSH assay uses two monoclonal anti-TSH antibodies recognizing different determinants of the thyroid-stimulating hormone molecule. Typically, the serum TSH level in thyrotoxic persons is suppressed to 0.05 μU per ml even if clinical signs of disease are mild. The physiologically hyperthyroid state can be diagnosed with assurance even when a patient is asymptomatic; not infrequently, this occurs in a hyperthyroid person taking antithyroid drugs, in hypothyroid patients taking levothyroxine sodium, or in a patient with a thyroid nodule or with surgically treated thyroid carcinoma who is on thyroxine-replacement therapy at a dose designed deliberately to suppress maximally the serum TSH level. Persistently suppressed levels of TSH during treatment with antithyroid drugs indicate that the treatment is inadequate. A fully suppressed serum TSH level correlates well with the failure of serum TSH to rise after administration of thyrotropin-releasing hormone, so the latter test should rarely be needed to confirm thyrotoxicosis or to establish that suppressive thyroxine treatment is adequate in patients with treated thyroid carcinoma.

A parallel measurement of the serum free thyroxine level is usually recommended to assess the biochemical magnitude of hyperthyroidism. Obvious clinical toxicity is usually associated with values well above the normal range; however, mild hyperthyroidism, as defined by serum TSH suppression, is not infrequently associated with free thyroxine levels in the upper portion (1.6 to 2.3 ng per dl) of the normal range (0.8 to 2.3 ng per dl). Critically ill patients with nonthyroidal illness who are not receiving dopamine have normal pituitary-thyroid function.

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Dipyridamole Myocardial Perfusion Scintigraphy

MYOCARDIAL PERFUSION SCINTIGRAPHY using thallium 201 during dynamic exercise is now well established as an important clinical aid for the diagnosis and prognosis of coronary artery disease. A large fraction of patients, a third or more, with known or suspected coronary disease are ill, debilitated, or timid, however, and cannot undergo dynamic exercise stress testing. Others, such as patients after myocardial infarction, should not be or generally are not submitted to a maximal exercise effort. Nevertheless, symptoms in these patients are often atypical or absent despite the fact that they may harbor the most severe, life-threatening disease. Compounding the problem is the fact that many of them often face a significant surgical intervention. Patients with peripheral vascular disease claudicate, and their limited activities prevent the early appearance of cardiac symptoms. Yet, their major morbidity and mortality relate to associated, frequently occult, coronary disease, which is the leading cause of postoperative complications.

Dipyridamole inhibits circulating adenosine deaminase, resulting in reduced degradation and increased levels of the potent arteriolar dilator, adenosine. Administered intravenously, the agent has significant effects on the coronary resistance vessels, the small arterioles, resulting in a threefold to fivefold increment in coronary artery flow through normal vessels. This flow augmentation is limited by a fixed stenosis, however. Animal experiments indicate differential augmentation of coronary perfusion in both normal and stenotic vessels. Coronary flow demands, however, are generally not increased and ischemia is rarely documented, possibly due to a "steal" effect or to an actual flow reduction.

A saline solution containing 0.56 mg per kg of dipyridamole is infused over four minutes with full symptomatic and hemodynamic monitoring. Ingestion of a dipyridamole slurry has also been reported to be effective. Thallium is administered at seven minutes, and images are acquired shortly thereafter and four hours later at redistribution, when basal flow distribution of the radionuclide is restored. Uniform flow augmentation in the absence of significant coronary lesions produces a homogeneous radionuclide distribution and a normal image. The presence of pathophysiologically signifi-